Title
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USEFULNESS OF INTRAOPERATIVE ESOPHAGOGASTRODUODENOSCOPY IN A PATIENT WITH LUPUS ENTERITIS

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Abstract: A 19-year-old Japanese woman had melena 2 months after systemic lupus erythematosus was diagnosed. Colonoscopy showed diffuse ulceration with bleeding in the ileum, suggesting that the melena was due to ischemic enteritis associated with lupus enteritis. Because treatment with high doses of steroid, anticoagulants, and cyclophosphamide pulse was ineffective, surgical intervention was planned. On exploration, it was impossible to determine the extent of resection visually. Intraoperative esophagogastroduodenoscopy clearly revealed the border between the ulcer and normal area, permitting successful resection of the ileum and ileostomy. This is the first report to document the usefulness of esophagogastroduodenoscopy in surgical treatment of ischemic enteritis in a patient with systemic lupus erythematosus.

Key words: intraoperative esophagogastroduodenoscopy, lupus enteritis

INTRODUCTION

Lupus enteritis, especially ischemic enteritis, is one of the life-threatening complications in patients with systemic lupus erythematosus (SLE). Immediate surgical intervention is needed in patients with extensive bowel infarction, hemorrhage, or intestinal perforations. However, it is difficult to evaluate the condition precisely, and decide the timing of surgical intervention. We herein report a lupus
patient with ischemic enteritis, the first such patient, in whom intraoperative esophagastroduodenoscopy (EGDS) was useful in determining the extent of resection.

CASE REPORT

In August, 2000, a 19-year-old Japanese woman with vomiting, diarrhea, and melena was transferred to our hospital. In March, she was diagnosed as having SLE accompanied by malar rash, arthritis, proteinuria, leukopenia, positive anti-DNA antibodies, and a high titer of antinuclear antibodies, according to the 1982 American College of Rheumatology revised criteria for the classification of SLE. Initial treatment with prednisolone including pulse therapy alleviated malar rash and arthritis, and normalized the laboratory values. However, she had had vomiting, diarrhea, and melena since May, and those symptoms were refractory to the therapy.

On physical examination, the patient appeared seriously ill and thin. She had no clouding of consciousness or disorientation. The temperature was 37.0°C, pulse 135/min, and blood pressure 160/110 mmHg. The lungs were clear, and heart sounds normal. Abdominal examination revealed a fluid wave and increased bowel sounds. The extremities were markedly edematous, and generalized muscle atrophy was noted.

Laboratory investigations showed a high white blood cell count (16,600/mm³), thrombocytopenia (74,000/mm³), and anemia (RBC 3.42×10⁶/mm³, Hb 10.3 g/dl). Hypoproteinemia (3.1 g/dl) and hypoalbuminemia (1.8 g/dl) were remarkable. BUN and Cr were normal. The levels of CH50 and the titer of antinuclear antibodies were normal. The anticardiolipin antibody was negative. The urinalysis showed a slightly positive protein. Klebsiella pneumoniae and Enterococcus faecium were detected from stool culture.

Chest roentgenogram showed clouding of both lung fields. Abdominal roentgenogram showed air in the intestine but no free air or fluid levels. Ultrasound and computed tomographic scans of the abdomen showed massive ascites, thickening of the intestinal wall and dilated small intestine filled with fluid. Barium meal X-ray study showed shortening and effacement of mucosal folds at the small intestine. EGDS showed diffuse mucosal edema in the duodenum. Colonoscopy (CS) including terminal ileum examination showed edematous mucosa of the colon and diffuse ulceration with bleeding in the ileum. A mucosal cast desquamated from the ileum was collected during CS. Tc-99m albumin scintigraphy showed collection of tracer in the small intestine.

We concluded that her melena and hypoproteinemia were caused by lupus enteritis. Barium meal and CS findings suggested ischemic enteritis mainly in the ileum. She was placed on complete bowel rest and supported with aggressive fluid replacement via a central venous catheter. Treatment with high doses of steroid, anticoagulants, and cyclophosphamide pulse was initiated; however, her status was
Figure 1. A; Intraoperative esophagogastroduodenoscopic image showed normal mucosa with blood coming from the ulcers in the ileum. B; Diffuse ulceration was noted at 1 m from the Treitz ligament.

Figure 2. The resected intestine showed diffuse ulceration. The arrow indicates the border between the normal mucosa and ulcer at the oral side.

refractory. Surgical intervention was then planned to remove the ischemic ileum that caused severe hemorrhage and fluid loss.

On exploration, the small intestine was found to be markedly shortened and edematous; however, we were unable to identify the line of transection visually. Although CS was tried, the scope did not reach far enough. Finally EGDS (XQ–240, Olympus, Japan), which was performed with the aid of a surgeon, clearly revealed the border between the ulcer and normal area, which was located 100 cm from the Treitz ligament (Figure 1A and B). One hundred twenty centimeters of the ileum was resected; then, ileostomy was performed, and end of the ileum (10 cm from Bauhin's valve) was closed with a view to the future anastomosis.

The resected intestine showed diffuse ulceration (Figure 2). Pathological examination revealed ulcer and cell infiltration into the submucosa. Also, the arterioles in the submucosal layer were obstructed with fibrinoid degeneration,
which indicated ischemic enteritis (Figure 3).

Although her postoperative course was complicated by short bowel syndrome, she was discharged with fluid replacement via a central venous catheter.

DISCUSSION

Lupus enteritis is one of the most serious complications of SLE. The clinical and pathological spectrum of lupus enteritis varies widely from simple enteritis to ulceration, hemorrhage, infarction, and perforation\(^1\) \(^2\). The jejunum and ileum are the sites most commonly affected by lupus enteritis\(^3\). The underlying lesion in most of these conditions is vasculitis of smaller arteries or veins resulting from deposition of circulating immune complexes. Most patients with lupus enteritis are successfully treated with high doses of steroid, and if they are refractory to steroid therapy, cyclophosphamide or azathiopurine is added. Mesenteric or intestinal vasculitis is a life-threatening complication of lupus enteritis, usually associated with multisystem activity, which causes perforation and/or infarction in the intestine. Immediate surgical intervention is needed in patients with extensive bowel infarction, hemorrhage, or intestinal perforation.

The diagnosis of bowel ischemia is often difficult to make in lupus patients. CT scanning has been recommended for pre-operative evaluation\(^4\). Recently, exploratory laparoscopy has been proposed as one of the most useful examinations for diagnosis and evaluation in lupus enteritis\(^5\). We diagnosed this case as ischemic enteritis from the findings of CT scanning, CS, and barium meal X-ray study. Because her condition did not respond to the conventional therapy, surgical intervention was planned. Of the 3 methods to determine the extent of resection, open surgery and CS did not work; only EGDS helped.

Hiraishi et al. presented a patient and reviewed 10 with lupus enteritis ac-
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accompanied by massive and life-threatening hemorrhage\(^6\). In 6 of the 10 patients, the bleeding sites were identified by exploratory laparoscopy and angiography, and all 6 recovered from enteritis. The 3 patients in whom the bleeding sites were not identified died of gastrointestinal bleeding. The authors recommend early laparotomy to improve survival of lupus patients with gastrointestinal manifestations\(^5,6\). However, our experience with this patient indicates that laparoscopy is not always useful in disclosing lesions or determining an extent of resection.

In conclusion, we recommend intraoperative EGDS in patients with lupus enteritis in whom it is difficult to determine an extent of resection.

REFERENCES